? b 155
11oct02 13:50:59 User208669 Session D2132.1
$0.29 0.082 DialUnits File1
$0.29 Estimated cost File1
$0.01 TELNET
$0.30 Estimated cost this search
$0.30 Estimated total session cost 0.082 DialUnits

File 155:MEDLINE(R) 1966-2002/Oct W1
*File 155: Alert feature enhanced for multiple files, duplicates removal, customized scheduling. See HELP ALERT.

? ds

Set	Items	Description
S1	2776	SENDAI
S2	30671	CHIMER? OR CHIMAER?
S3	34	S1 AND S2
S4	8812819	PY <1996
S5	23	S3 AND S4
S6	11	S3 NOT S5
S7	246947	VARIANT? OR MUTANT?
S8	232	S1 AND S7
S9	83591	VARIANT?
S10	46	S1 AND S9
S11	261279	MUTATION?
S12	166	S1 AND S11
S13	52	S7(3N)S1 NOT S10

? t s5/7/1 11

5/7/1
DIALOG(R)File 155:MEDLINE(R)
08728935 96074511 PMID: 7491760
Mutations in conserved domain I of the Sendai virus L polymerase protein uncouple transcription and replication.
Chandrika R; Horikami S M; Smallwood S; Moyer S A
Department of Molecular Genetics, University of Florida College of Medicine, Gainesville 32610-0266, USA.
Virology (UNITED STATES) Nov 10 1995, 213 (2) p352-63, ISSN 0042-6822 Journal Code: 0110674
Contract/Grant No.: AI14594; AI; NIAID
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
To begin to map functional domains of the Sendai P-L RNA polymerase complex we wanted to characterize the P binding site on the Sendai L protein. Analysis of in vitro and in vivo P-L polymerase complex formation with carboxyl-truncations of the L protein showed that the N-terminal half of the protein was required. Site-directed mutagenesis of the Sendai virus L gene was employed to change amino acids within a highly conserved region of the N-terminal domain I from amino acids (aa) 348-379 singly or in pairs from the Sendai to the corresponding measles L sequence or to alanine. The mutant L proteins coexpressed with the viral P and NP proteins in mammalian cells were assayed for their ability to form the P-L complex and to synthesize RNA in vitro and showed a variety of defective phenotypes. While most of the mutant L proteins still formed the P-L polymerase complex, a change from serine to arginine at aa 368 and a three-amino-acid insertion at aa 379 virtually abolished both complex formation and RNA synthesis. Changes of aas 370 and 376-377 in the L protein gave only small decreases in viral RNA synthesis. Substitutions at either aas 349-350 or aas 354-355 and a three-amino-acid insertion at aa 348 in the L protein yielded enzymes that catalyzed significant transcription, but were defective in DI RNA replication, thus differentially affecting the two processes. Since DI leader RNA, but not genome RNA, was still synthesized by this class of mutants, the defect in replication appears to be in the ability of the mutant enzyme to package newly synthesized nascent RNA. Single changes at aas 362, 363, and 366 in the L protein gave enzymes with severely decreased overall RNA synthesis, although some leader RNA was synthesized, suggesting that they cannot transcribe or replicate past the leader gene. These studies have identified a region in conserved domain I critical for multiple functions of the Sendai virus L protein.
Record Date Created: 19960102

5/7/11
DIALOG(R)File 155:MEDLINE(R)
06519879 90218050 PMID: 2157809
Localization of P protein binding sites on the Sendai virus nucleocapsid.
Ryan K W; Murti K G; Portner A
Department of Virology and Molecular Biology, St Jude Children's Research Hospital, Memphis, Tennessee 38101.
Journal of general virology (ENGLAND) Apr 1990, 71 (Pt 4) p997-1000, ISSN 0022-1317 Journal Code: 0077340
Contract/Grant No.: AI05343; AI; NIAID; CA21765; CA; NCI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Previous studies have shown that the molecules of P protein associated with transcriptionally active Sendai virus nucleocapsids are arranged in discrete clusters. Our study investigates whether or not this localized distribution is due to the existence of only a few P protein binding sites on the nucleocapsid core. We used immunoelectron microscopy to examine whether additional P proteins could bind at locations between the groups of

endogenous P proteins. To differentiate between endogenous and added proteins, we constructed a recombinant gene which instructs the in vitro synthesis of a chimeric protein containing the carboxyl-terminal nucleocapsid-binding region of P protein, fused to chloramphenicol acetyltransferase (CAT). Immunogold labelling, using an antibody to the CAT moiety, revealed at the electron microscope level, that the chimeric product bound to nucleocapsids at many sites located over the entire length of the nucleocapsid. This indicated that the localized distribution of P protein molecules is not due to a limited number of P protein binding sites on the nucleocapsid core.
Record Date Created: 19900521

? t s10/7/20 25 28 33 36 37 38 39 41 43

10/7/20
DIALOG(R)File 155:MEDLINE(R)
06805421 91112000 PMID: 1846501
Identification of amino acid positions associated with neuraminidase activity of the hemagglutinin-neuraminidase glycoprotein of Sendai virus.
Gorman W L; Takahashi T; Scroggs R A; Portner A
Department of Virology and Molecular Biology, St. Jude Children's Research Hospital, Memphis, Tennessee 38101.
Virology (UNITED STATES) Feb 1991, 180 (2) p803-8, ISSN 0042-6822 Journal Code: 0110674
Contract/Grant No.: AI11949; AI; NIAID; CA21765; CA; NCI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Identification of amino acid positions associated with neuraminidase activity on the hemagglutinin-neuraminidase (HN) glycoprotein of paramyxoviruses has been difficult because neuraminidase-inhibiting antibodies are not neutralizing and thus, escape mutants have not been isolated. Instead, many investigators have correlated an altered neuraminidase (NA) activity of natural virus variants, such as plaque-size variants, with sequence changes in the HN protein. To identify regions on the HN glycoprotein of Sendai virus (SV) that are associated with NA activity, we investigated NA activity of three plaque-size variants which potentially differed from the standard SV (SV/std). NA activity was measured by the ability of virus to elute from chicken erythrocytes as a result of cleaving sialic acid receptors, and by the ability of virus to cleave sialic acid from the small trisaccharide neuraminlactose and the larger substrate fetuin in an in vitro assay. Virions purified from each of the isolated plaques had a HN content and hemagglutinating activity similar to that of SV/std, yet each variant eluted much more rapidly from chicken erythrocytes than SV/std. In vitro NA activity of the plaque-size variants was 1.6 to 3.8 times greater than that of SV/std, providing supporting evidence for the elution data. Although all plaque-size variants showed elevated NA activity, there was no correlation of activity with plaque size. Sequence analysis showed that one of the variants had an amino acid change from glutamic acid to valine at position 165 and from lysine to glutamic acid at position 461, while a second variant had only the change at position 461. A third variant had a nearby change at position 468, from threonine to lysine. Taken together, these data support the conclusion that the amino acid residues at positions 461-468 and 165 are involved in neuraminidase activity of SV.
Record Date Created: 19910222

10/7/25
DIALOG(R)File 155:MEDLINE(R)
05875033 88306249 PMID: 2841801
Characterization of a pantropic variant of Sendai virus derived from a host range mutant.
Tashiro M; Pritzer E; Khoshnan M A; Yamakawa M; Kuroda K; Klenk H D; Rott R; Seto J T
Institut fur Virologie, Justus-Liebig-Universitat Giessen, Federal Republic of Germany.
Virology (UNITED STATES) Aug 1988, 165 (2) p577-83, ISSN 0042-6822 Journal Code: 0110674
Contract/Grant No.: AI-24096; AI; NIAID; RR 081010-16; RR; NCRR
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
A variant (F1-R) was isolated from a temperature-sensitive host range mutant (ts-f1) of Sendai virus. F1-R was no longer temperature-sensitive but it retained the host range phenotype. Unlike wild-type virus, F1-R and ts-f1 undergo multiple cycles of replication in several cell lines in the absence of trypsin. This was attributed to proteolytic activation of the fusion (F) glycoprotein of the host range mutants, in cell nonpermissive to wild-type virus. In mice infected intranasally the variant F1-R caused a generalized infection. This was shown by immunohistology and with infectious virus being recovered from several organs whereas infection with wild-type virus was restricted to the lung. These observations indicate that the pantropic property of F1-R is the result of proteolytic activation of the virus by ubiquitous proteases. Nucleotide sequence analyses revealed that ts-f1 and F1-R differed from the wild-type virus by mutations at the region of the cleavage site of F and at the glycosylation site of the F2 subunit. The findings indicated that these mutations are responsible for the increased cleavability of the F protein of ts-f1 and F1-R and therefore are important determinants for the pantropism of F1-R.
Record Date Created: 19880916

10/7/28
DIALOG(R)File 155:MEDLINE(R)
05396922 87151157 PMID: 2435061

The fusion glycoprotein of Sendai virus: sequence analysis of an epitope involved in fusion and virus neutralization.
Portner A; Scroggs R A; Naeve C W
Virology (UNITED STATES) Apr 1987, 157 (2) p556-9, ISSN 0042-6822 Journal Code: 0110674
Contract/Grant No.: AI 11949; AI; NIAID; CA 21765; CA; NCI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
To localize the amino acid residues on the F glycoprotein that are involved in Sendai virus fusion and virus neutralization, an anti-F monoclonal antibody which inhibits these functions was used to select three antigenic variants. Sequence analysis of the entire F gene of the three variants identified a single mutation that was responsible for the loss of antibody binding. The mutation, a proline to glutamine substitution at residue 399, was at a position in the primary sequence far removed from the hydrophobic F1-NH2 terminus thought to be directly involved in fusion. A synthetic peptide, comprising amino acid sequences in the region of the mutation, bound to the antibody used to select the variants, suggesting that the site of mutation is also the site of antibody binding. This information suggests that in the three-dimensional structure of the F molecule the amino acid residues around proline 399 are located close to the F1-NH2 terminus, and that fusion is directly inhibited by antibody binding. Other less likely alternatives are discussed.
Record Date Created: 19870422

10/7/33
DIALOG(R)File 155:MEDLINE(R)
04426805 84113530 PMID: 6198445
Antigenic variation of HVJ (Sendai virus) HN glycoprotein detectable by monoclonal antibodies during persistent infection.
Sato H; Ogura H; Tanaka J; Hatano M
Journal of general virology (ENGLAND) Jan 1984, 65 (Pt 1) p185-9, ISSN 0022-1317 Journal Code: 0077340
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Three newly established monoclonal hybridoma antibodies to the haemagglutinin molecule of HVJ, designated A7, B3 and F11, recognize operationally non-overlapping antigenic determinants and have neutralizing activity. Using these antibodies, the frequencies of occurrence of neutralization-resistant antigenic variants were analysed in virus populations released from four cell lines persistently infected with HVJ, namely GM2-HVJ, LLCMK2-HVJ, Vero-HVJ and GEsl-HVJ at various passage stages. Antigenic variants were selected from culture fluids of these HVJ carrier cells at a total frequency of 10(-3.3), 10(-3.8) and 10(-3.6) by monoclonal antibodies A7, B3 and F11, respectively. These values were considerably higher than those of 10(-4.7) to 10(-5.2) detected in a stock preparation of wild-type virus with these antibodies. All the variant viruses isolated as above were negative in neutralization, haemagglutination inhibition and immunofluorescent staining tests with each monoclonal antibody used for their isolation, but were positive with the other antibodies.
Record Date Created: 19840301

10/7/36
DIALOG(R)File 155:MEDLINE(R)
03802850 82065148 PMID: 6118055 N05
Comparison of the protein composition of two plaque variants of Sendai virus.
Sugita K
Acta virologica. English ed (CZECHOSLOVAKIA) Sep 1981, 25 (5) p330-3, ISSN 0001-723X Journal Code: 0370401
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
The protein composition of plaque variants of Sendai virus were compared by slab polyacrylamide gel electrophoresis. The RL and RS variants displayed clearly different P-protein mobilities; the P protein of the RS variant migrated faster than that of the RL variant, while no difference was found with proteins L, HANA, NP, F1 and M. These protein patterns differed from those of the Fushimi and Z strains.
Record Date Created: 19820120

10/7/37
DIALOG(R)File 155:MEDLINE(R)
03691103 81244279 PMID: 6265739
Biological properties of plaque-size variants of Sendai virus.
Sugita K; Maru M; Sato K
Microbiology and immunology (JAPAN) 1981, 25 (4) p353-60, ISSN 0385-5600 Journal Code: 7703966
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Large (RL)-and small (RS)-plaque variants of Sendai virus were isolated in culture of LLCMK2 cells in the presence of trypsin and their biological properties were determined. The RL variant was more virulent to mice than the RS variant. The RL variant had a higher growth rate than the RS variant in multiple-step growth in the presence of trypsin, but the two variants had an almost equal growth rate in its absence. Restoration of hemolytic

activity in cleavage of the F protein of the RL variant were achieved by milder trypsin treatment than was needed for the RS variant.
Record Date Created: 19810925

10/7/38
DIALOG(R)File 155:MEDLINE(R)
03441589 80237616 PMID: 6249026
Similar frequencies of antigenic variants in Sendai, vesicular stomatitis, and influenza A viruses.
Portner A; Webster R G; Bean W J
Virology (UNITED STATES) Jul 15 1980, 104 (1) p235-8, ISSN 0042-6822 Journal Code: 0110674
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Record Date Created: 19800928

10/7/39
DIALOG(R)File 155:MEDLINE(R)
03361501 80184338 PMID: 232513
[Plaque formation of Sendai virus in a variant isolated from the colony of BHK21/WI-2 cells formed in soft agar or in BHK21/WI-2 cells infected with Rous sarcoma virus.]
Sato M; Maeda N; Yoshida H; Shirasuna K; Yanagawa T; Kubo K; Miyazaki T
Journal of Osaka University Dental School (JAPAN) Dec 1978, 18 p11-8, ISSN 0473-4599 Journal Code: 7503132
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Record Date Created: 19800726

10/7/41
DIALOG(R)File 155:MEDLINE(R)
03193297 80010991 PMID: 481258
Biochemical characterization of H2S-positive Salmonella sendai strains isolated in Hong Kong.
Chau P Y; Huang C T
Microbiology and immunology (JAPAN) 1979, 23 (3) p125-9, ISSN 0385-5600 Journal Code: 7703966
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
The biochemical properties of 8 H2S-positive variant strains of Salmonella sendai isolated from patients and carriers in Hong Kong were studied. Apart from the production of H2S, all these strains showed typical properties of S. sendai and a dependence on tryptophan. Their capacity to utilize different forms of sulphur sources varied, ranging from being capable of utilizing SO4, SO3, S2O3 and cystein to only cystein as the sulphur source.
Record Date Created: 19791128

10/7/43
DIALOG(R)File 155:MEDLINE(R)
02974096 79041149 PMID: 213044
Recovery of a Sendai virus variant with temperature-sensitive hemolytic activity from persistently infected cells from mouse brain.
Collins A R; Flanagan T D
Archives of virology (AUSTRIA) 1978, 58 (2) p81-93, ISSN 0304-8608 Journal Code: 7506870
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
A persistently infected cell line designated MB/Senas was established by cultivation of mouse brain cells from four-day-old C3H mice infected intracerebrally at birth with 10(6) PFU of Sendai virus, strain 52. After 5 passages, 0.16 per cent of Sendai52 antiserum (containing two 50 per cent plaque reducing doses/ml of serum) was introduced into the culture medium. The addition of antiserum was accompanied by a rise in cell-associated viral antigen from a level of 5 per cent antigen positive cells to 100 per cent demonstrable by both intracellular and membrane immunofluorescence. A variant of Sendai52 virus, designated Sendaias, was recovered from MB/Senas by inoculation of supernatant medium into chick embryos. Infection of chick embryos at 37 degrees C was abortive. Fifty per cent or less of chick embryos infected at dilutions 10(-1) to 10(-9) yielded detectable virus. Hemagglutination (HA) was weak but could be improved by trypsinization of allantoic fluids. Neuraminidase (NA) activity was barely detectable. Hemolysis (HE) was absent. Propagation of Sendaias virus at 33 degrees C showed no change from weak HA and NA activities but HE activity was now apparent which was temperature sensitive. Mortality of infected chick embryos increased to 100 per cent. HE activity and lethality for chick embryos was thermolabile at 45 degrees C.
Record Date Created: 19781220
TIMEOUT: Logged Off 10/11/02 14:12:27 by System

Connection closed by remote host
Reconnected in file 155 11oct02 14:32:32
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File 155:MEDLINE(R) 1966-2002/Oct W1
*File 155: Alert feature enhanced for multiple files, duplicates

removal, customized scheduling. See HELP ALERT.

Set Items Description
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Cost is in DialUnits
? ds
Set Items Description
S1 2776 SENDAI
S2 30671 CHIMER? OR CHIMAER?
S3 34 S1 AND S2
S4 8812819 PY <1996
S5 23 S3 AND S4
S6 11 S3 NOT S5
S7 246947 VARIANT? OR MUTANT?
S8 232 S1 AND S7
S9 83591 VARIANT?
S10 46 S1 AND S9
S11 261279 MUTATION?
S12 166 S1 AND S11
S13 52 S7(3N)S1 NOT S10
? t s13/7/14 14 16-19 21-24 27 37
13/7/14
DIALOG(R)File 155:MEDLINE(R)
09628920 98062143 PMID: 9400971
Isolation of an avirulent mutant of Sendai virus with two amino acid mutations from a highly virulent field strain through adaptation to LLC-MK2 cells.
Itoh M; Isegawa Y; Hotta H; Homma M
Department of Microbiology, Kobe University School of Medicine, Japan. masae@med.kobe-u.ac.jp
Journal of general virology (ENGLAND) Dec 1997, 78 (Pt 12) p3207-15, ISSN 0022-1317 Journal Code: 0077340
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
A field strain of Sendai virus (SeV) Ohita-M1 (M1) was isolated from an epidemic in an animal laboratory by passaging in mice. A mutant strain, Ohita-MVC11 (MVC11), was then obtained by passaging M1 in rhesus monkey (LLC-MK2) cells. MVC11 was adapted to LLC-MK2 cells and produced 20 times higher levels of infectious virus than M1. This increased production of infectious virus in LLC-MK2 cells was associated with enhanced viral gene expression. However, MVC11 could not replicate efficiently in mouse lung and was not lethal to mice even when inoculated at a titre of 8 x 10(5) cell-infecting units (CIU) per mouse. On the other hand, with an inoculum of only 4 x 10(1) CIU per mouse, corresponding to 1 LD50, M1 replicated well in mouse lung and was highly virulent to mice. Nucleotide and deduced amino acid sequence analyses of the entire genomes of M1 and MVC11 revealed that adaptation to LLC-MK2 cells and the attenuation of mouse pathogenicity of MVC11 were associated with only two amino acid substitutions; one on the C protein (Phe substituted by Ser at position 170) and the other on the RNA polymerase, the L protein (Glu substituted by Ala at position 2050).
Record Date Created: 19980105

13/7/14
DIALOG(R)File 155:MEDLINE(R)
09628920 98062143 PMID: 9400971
Isolation of an avirulent mutant of Sendai virus with two amino acid mutations from a highly virulent field strain through adaptation to LLC-MK2 cells.
Itoh M; Isegawa Y; Hotta H; Homma M
Department of Microbiology, Kobe University School of Medicine, Japan. masae@med.kobe-u.ac.jp
Journal of general virology (ENGLAND) Dec 1997, 78 (Pt 12) p3207-15, ISSN 0022-1317 Journal Code: 0077340
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
A field strain of Sendai virus (SeV) Ohita-M1 (M1) was isolated from an epidemic in an animal laboratory by passaging in mice. A mutant strain, Ohita-MVC11 (MVC11), was then obtained by passaging M1 in rhesus monkey (LLC-MK2) cells. MVC11 was adapted to LLC-MK2 cells and produced 20 times higher levels of infectious virus than M1. This increased production of infectious virus in LLC-MK2 cells was associated with enhanced viral gene expression. However, MVC11 could not replicate efficiently in mouse lung and was not lethal to mice even when inoculated at a titre of 8 x 10(5) cell-infecting units (CIU) per mouse. On the other hand, with an inoculum of only 4 x 10(1) CIU per mouse, corresponding to 1 LD50, M1 replicated well in mouse lung and was highly virulent to mice. Nucleotide and deduced amino acid sequence analyses of the entire genomes of M1 and MVC11 revealed that adaptation to LLC-MK2 cells and the attenuation of mouse pathogenicity of MVC11 were associated with only two amino acid substitutions; one on the C protein (Phe substituted by Ser at position 170) and the other on the RNA polymerase, the L protein (Glu substituted by Ala at position 2050).
Record Date Created: 19980105

13/7/16
DIALOG(R)File 155:MEDLINE(R)
08961706 96323115 PMID: 8709221
Involvement of the mutated M protein in altered budding polarity of a pantropic mutant, F1-R, of Sendai virus.
Tashiro M; McQueen N L; Seto J T; Klenk H D; Rott R
Department of Virology 1, National Institute of Health, Tokyo, Japan.

mtashiro@nih.go.jp
Journal of virology (UNITED STATES) Sep 1996, 70 (9) p5990-7, ISSN 0022-538X Journal Code: 0113724
Contract/Grant No.: AI 132677-01A1; AI; NIAID; AI 31191-01; AI; NIAID; SO6 GM08 101-22; GM; NIGMS
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Wild-type Sendai virus buds at the apical plasma membrane domain of polarized epithelial MDCK cells, whereas a pantropic mutant, F1-R, buds at both the apical and basolateral domains. In F1-R-infected cells, polarized protein transport and the microtubule network are impaired. It has been suggested that the mutated F and/or M proteins in F1-R are responsible for these changes (M. Tashiro, J. T. Seto, H.-D. Klenk, and R. Rott, J. Virol. 67:5902-5910, 1993). To clarify which gene or mutation(s) was responsible for the microtubule disruption which leads to altered budding of F1-R, MDCK cell lines containing the M gene of either the wild type or F1-R were established. When wild-type M protein was expressed at a level corresponding to that synthesized in virus-infected cells, cellular polarity and the integrity of the microtubules were affected to some extent. On the other hand, expression of the mutated F1-R M protein resulted in the formation of giant cells about 40 times larger than normal MDCK cells. Under these conditions, the effects on the microtubule network were enhanced. The microtubules were disrupted and polarized protein transport was impaired as indicated by the nonpolarized secretion of gp80, a host cell glycoprotein normally secreted from the apical domain, and bipolar budding of wild-type and F1-R Sendai viruses. The mutated F glycoprotein of F1-R was transported bipolarly in cells expressing the F1-R M protein, whereas it was transported predominantly to the apical domain when expressed alone or in cells coexpressing the wild-type M protein. These findings indicate that the M protein of F1-R is involved in the disruption of the microtubular network, leading to impairment of cellular polarity, bipolar transport of the F glycoprotein, and bipolar budding of the virus.
Record Date Created: 19960910

13/7/17
DIALOG(R)File 155:MEDLINE(R)
07875094 94012782 PMID: 8408048
Temperature-sensitive phenotype of a mutant Sendai virus strain is caused by its insufficient accumulation of the M protein.
Kondo T; Yoshida T; Miura N; Nakanishi M
Institute for Molecular and Cellular Biology, Osaka University, Japan.
Journal of biological chemistry (UNITED STATES) Oct 15 1993, 268 (29) p21924-30, ISSN 0021-9258 Journal Code: 2985121R
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
We investigated the process interrupting the production of a temperature-sensitive mutant strain of Sendai virus, Cl.151, at the nonpermissive temperature (38 degrees C). The amount of virus M protein increased up to 6-fold when the cells persistently infected with Cl.151 strain at 38 degrees C are transferred to 32 degrees C, while the amount of nucleocapsid proteins did not alter. Cl.151 strain could restore virus production at 38 degrees C not only by the supplementation of M protein of wild type (Z) strain but also by the supplementation of M protein of Cl.151 strain. Neither the amount of M mRNA nor the rate of synthesis of M protein was altered by temperature in cells infected with the Cl.151 strain. However, we found that M protein of Cl.151 virus, which has 3-amino acid alterations from the wild type, was highly unstable at 38 degrees C when expressed under the control of an actin promoter. These results clearly show that Sendai virus M protein has a critical role in the production of virus particles without affecting virus gene expression.
Record Date Created: 19931118

13/7/18
DIALOG(R)File 155:MEDLINE(R)
07850180 93381791 PMID: 8396659
Possible involvement of microtubule disruption in bipolar budding of a Sendai virus mutant, F1-R, in epithelial MDCK cells.
Tashiro M; Seto J T; Klenk H D; Rott R
Department of Virology, Jichi Medical School, Tochigi-ken, Japan.
Journal of virology (UNITED STATES) Oct 1993, 67 (10) p5902-10, ISSN 0022-538X Journal Code: 0113724
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Envelope glycoproteins F and HN of wild-type Sendai virus are transported to the apical plasma membrane domain of polarized epithelial MDCK cells, where budding of progeny virus occurs. On the other hand, a pantropic mutant, F1-R, buds bipolarly at both the apical and basolateral domains, and the viral glycoproteins have also been shown to be transported to both of these domains (M. Tashiro, M. Yamakawa, K. Tobita, H.-D. Klenk, R. Rott, and J.T. Seto, J. Virol. 64:4672-4677, 1990). MDCK cells were infected with wild-type virus and treated with the microtubule-depolymerizing drugs colchicine and nocodazole. Budding of the virus and surface expression of the glycoproteins were found to occur in a nonpolarized fashion similar to that found in cells infected with F1-R. In uninfected cells, the drugs were shown to interfere with apical transport of a secretory cellular glycoprotein, gp80, and basolateral uptake of [35S]methionine as well as to disrupt microtubule structure, indicating that cellular polarity of MDCK

cells depends on the presence of intact microtubules. Infection by the F1-R mutant partially affected the transport of gp80, uptake of [35S]methionine, and the microtubule network, whereas wild-type virus had a marginal effect. These results suggest that apical transport of the glycoproteins of wild-type Sendai virus in MDCK cells depends on intact microtubules and that bipolar budding by F1-R is possibly due, at least in part, to the disruption of microtubules. Nucleotide sequence analyses of the viral genes suggest that the mutated M protein of F1-R might be involved in the alteration of microtubules.
Record Date Created: 19931012

13/7/19
DIALOG(R)File 155:MEDLINE(R)
07367483 92300359 PMID: 1339465
Changes in specific cleavability of the Sendai virus fusion protein: implications for pathogenicity in mice.
Tashiro M; Seto J T; Choosakul S; Hegemann H; Klenk H D; Rott R
Department of Virology, Jichi Medical School, Tochigi, Japan.
Journal of general virology (ENGLAND) Jun 1992, 73 (Pt 6) p1575-9, ISSN 0022-1317 Journal Code: 0077340
Contract/Grant No.: RR 08101-18; RR; NCRR
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Sendai virus mutants, KDe-21 and KDe-62, which had undergone multiple cycles of replication in Madin Darby canine kidney (MDCK) cells in the absence of exogenous proteases were isolated. The fusion (F) protein of the mutants regained proteolytic cleavability in MDCK cells and chick embryos, but the F protein remained non-cleavable in other cell lines. Unlike the F protein of wild-type (wt) virus, the mutant F was resistant to trypsin but was sensitive to elastase and, to a lesser extent, to chymotrypsin. Sequence analyses of the F gene and the F protein revealed an amino acid substitution at the cleavage site, Arg(116) to Ile, which conferred trypsin resistance and enhanced cleavability at Ile(116) by elastase and host proteases present in MDCK cells and in chicken embryos. In contrast to the pneumopathogenicity in mice of wt Sendai virus, the KDe mutants were non-pathogenic; cleavage activation of the F protein did not occur in the lungs and thereby infection was terminated after an initial cycle of replication.
Record Date Created: 19920723

13/7/21
DIALOG(R)File 155:MEDLINE(R)
07026701 91335752 PMID: 1651590
Pneumotropic revertants derived from a pantropic mutant, F1-R, of Sendai virus.
Tashiro M; James I; Karri S; Wahn K; Tobita K; Klenk H D; Rott R; Seto J T
Department of Virology, Jichi Medical School, Tochigi, Japan.
Virology (UNITED STATES) Sep 1991, 184 (1) p227-34, ISSN 0042-6822 Journal Code: 0110674
Contract/Grant No.: RR 08101-18; RR; NCRR
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Revertants were isolated from the protease activation mutant of Sendai virus, F1-R, which causes a systemic infection in mice. The fusion (F) glycoprotein of F1-R is susceptible to activation cleavage by ubiquitous cellular proteases and is thus responsible for pantropism in mice (Tashiro et al., 1988. Virology 165, 577-583). The revertants regained several phenotypes of wild-type virus; they required exogenous trypsin for activation of the F protein in cell cultures and in nonpulmonary mouse tissues and they were exclusively pneumotropic in mice. On the other hand, phenotypes of F1-R that remained unchanged by the revertants were bipolar budding in polarized epithelial cells, enhanced electrophoretic migration of the matrix protein, and the lack of a glycosylation site in the F2 subunit of the F protein. Comparative RNA sequence analysis of the F gene of the revertants revealed that the reduced cleavability of the F protein of the revertants was the result of the predicted single amino acid reversion (Pro to Ser) at residue 115 adjacent to the cleavage site. Thus the sequence at the cleavage site of the revertants was Ser-Lys compared with Pro-Lys for F1-R and Ser-Arg for wild-type virus. The results indicate that enhanced cleavability of the glycoprotein, a feature often associated with multiple basic residues within the cleavage site of paramyxovirus F proteins and influenza virus hemagglutinins, can also be determined by a single basic amino acid following proline. Additionally, the revertants were less susceptible to the activator for wild-type virus present in mouse lungs and less pathogenic for this organ than wild-type virus. These results provide further evidence that proteolytic activation of the F protein by host proteases is the primary determinant for organ tropism and pathogenicity of Sendai virus in mice. One of the revertants was also temperature sensitive (ts); the ts lesion in the nucleoprotein gene was identical to that found in ts-f1, the ts host range mutant from which F1-R was derived.
Record Date Created: 19910918

13/7/22
DIALOG(R)File 155:MEDLINE(R)
07001282 91311425 PMID: 1649904
The major glycoprotein of Sendai virus is dispensable for efficient virus particle budding.
Stricker R; Roux L

Department of Microbiology, University of Geneva, Medical School, Switzerland.
Journal of general virology (ENGLAND) Jul 1991, 72 (Pt 7) p1703-7, ISSN 0022-1317 Journal Code: 0077340
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
A temperature-sensitive mutant of Sendai virus, ts271, when grown at restrictive temperature is known to produce virions lacking integral haemagglutinin-neuraminidase (HN). In this study, it is shown that the transmembrane-cytoplasmic tail of HN is not detected either. This apparent complete lack of HN does not affect budding efficiency.
Record Date Created: 19910827

13/7/23
DIALOG(R)File 155:MEDLINE(R)
06701149 91012818 PMID: 2170692
Pneumopathogenicity of a Sendai virus protease-activation mutant, TCs, which is sensitive to trypsin and chymotrypsin.
Itoh M; Ming T D; Hayashi T; Mochizuki Y; Homma M
Department of Microbiology, Kobe University School of Medicine, Japan.
Journal of virology (UNITED STATES) Nov 1990, 64 (11) p5660-4, ISSN 0022-538X Journal Code: 0113724
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
A protease-activation mutant of Sendai virus, TCs, was isolated from a trypsin-resistant mutant, TR-5. TCs was activated in vitro by both trypsin and chymotrypsin. TCs was, however, less sensitive to trypsin and chymotrypsin than were the wild-type virus and TR-5, respectively. F protein of TCs had a single amino acid substitution at residue 114 from glutamine to arginine, resulting in the appearance of the new cleavage site for trypsin and the shift of the cleavage site for chymotrypsin. Activation of TCs in the lungs of mice occurred less efficiently than that of the wild type, and TCs caused a less severe pneumopathogenicity than did the wild-type virus, which supports our previous view that the in vitro trypsin sensitivity of Sendai virus can be a good indication of pneumopathogenicity in mice.
Record Date Created: 19901115

13/7/24
DIALOG(R)File 155:MEDLINE(R)
06677163 90376416 PMID: 2168957
Altered budding site of a pantropic mutant of Sendai virus, F1-R, in polarized epithelial cells.
Tashiro M; Yamakawa M; Tobita K; Seto J T; Klenk H D; Rott R
Department of Virology, Jichi Medical School, Tochigi, Japan.
Journal of virology (UNITED STATES) Oct 1990, 64 (10) p4672-7, ISSN 0022-538X Journal Code: 0113724
Contract/Grant No.: RR 08101-18; RR; NCRR
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
A protease activation mutant of Sendai virus, F1-R, causes a systemic infection in mice, whereas wild-type virus is exclusively pneumotropic (M. Tashiro, E. Pritzer, M. A. Khoshnan, M. Yamakawa, K. Kuroda, H.-D. Klenk, R. Rott, and J. T. Seto, Virology 165:577-583, 1988). Budding of F1-R has been observed bidirectionally at the apical and basolateral surfaces of the bronchial epithelium of mice and of MDCK cells, whereas wild-type virus buds apically (M. Tashiro, M. Yamakawa, K. Tobita, H.-D. Klenk, R. Rott, and J. T. Seto, J. Virol. 64:3627-3634, 1990). In this study, wild-type virus was shown to be produced primarily from the apical site of polarized MDCK cells grown on permeable membrane filters. Surface immunofluorescence and immunoprecipitation analyses revealed that transmembrane glycoproteins HN and F were expressed predominantly at the apical domain of the plasma membrane. On the other hand, infectious progeny of F1-R was released from the apical and basolateral surfaces, and HN and F were expressed at both regions of the cells. Since F1-R has amino acid substitutions in F and M proteins but none in HN, the altered budding of the virus and transport of the envelope glycoproteins might be attributed to interactions by F and M proteins. These findings suggest that in addition to proteolytic activation of the F glycoprotein, the differential site of budding, at the primary target of infection, is a determinant for organ tropism of Sendai virus in mice.
Record Date Created: 19901017

13/7/27
DIALOG(R)File 155:MEDLINE(R)
06565100 90266486 PMID: 2161155
Nucleotide sequence analyses of the genes encoding the HN, M, NP, P, and L proteins of two host range mutants of Sendai virus.
Middleton Y; Tashiro M; Thai T; Oh J; Seymour J; Pritzer E; Klenk H D; Rott R; Seto J T
Department of Microbiology, California State University, Los Angeles 90032.
Virology (UNITED STATES) Jun 1990, 176 (2) p656-7, ISSN 0042-6822 Journal Code: 0110674
Contract/Grant No.: AI-24096; AI; NIAID; RR08101-18; RR; NCRR
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM

Record type: Completed
Comparative nucleotide sequence analyses of the genome of Sendai virus (strain Z) and two host range mutants, ts-f1 and F1-R, previously described revealed that the ts defect of ts-f1 can be attributed to two nucleotide exchanges in the NP gene. These exchanges lead to a single amino acid substitution. A single base pair change was found in both the P and L genes of F1-R, but not of ts-f1. Both host range mutants have the two same exchanges in the M gene. These additional mutations are discussed concerning their significance in the pantropic properties of the host range mutants.
Record Date Created: 19900705

13/7/37
DIALOG(R)File 155:MEDLINE(R)
05125596 86200404 PMID: 3009869
Nucleotide sequences that affect replicative and transcriptional efficiencies of Sendai virus deletion mutants.
Re G G; Kingsbury D W
Journal of virology (UNITED STATES) May 1986, 58 (2) p578-82, ISSN 0022-538X Journal Code: 0113724
Contract/Grant No.: AI 05343; AI; NIAID; CA 21765; CA; NCI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Structural features of the genomes of virus deletion mutants (DI virions) influence their replication efficiency. Among nonsegmented negative-strand RNA viruses, substitution of the genomic 3' terminus by a complementary copy of the 5' terminus (so-called "copy-back" sequence) could enhance replication either because the new 3' end is a better promoter of RNA replication or because DI RNAs that possess this sequence are incapable of acting as templates for transcription. Here we provide evidence that both mechanisms operate in mixed infections with Sendai virus DI RNAs. RNAs incapable of transcription always outgrew RNA species that were transcribed. This was true even when the 3'-terminal sequence of the untranscribed RNA was identical to the genomic 3' terminus, as in the case of an internally deleted DI genome (RNA Ra) rendered transcriptionally inert by point mutations of bases 47 and 51 at the 5' end of the positive-strand leader RNA template. Nevertheless, Ra was outgrown by a copy-back DI RNA, indicating that the 3' genomic end of Ra is a less efficient site for replication initiation than the copy-back sequence.
Record Date Created: 19860527
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File 155:MEDLINE(R) 1966-2002/Oct W2
*File 155: Alert feature enhanced for multiple files, duplicates
removal, customized scheduling. See HELP ALERT.

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S2        69   PY=1999 AND AU=SAKAI Y?
S3         2   SENDAI AND S2
S4       108   SENDAI AND (VECTOR? OR EXPRESSION(W)SYSTEM)
S5   1374088   PY>1999
S6        78   S4 NOT S5
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 1/3/1
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10396370   99388927   PMID: 10461831
 Sendai virus-based production of HIV type 1 subtype B and subtype E
envelope glycoprotein 120 antigens and their use for highly sensitive
detection of subtype-specific serum antibodies.
 Toriyoshi H; Shioda T; Sato H; Sakaguchi M; Eda Y; Tokiyoshi S; Kato K;
Nohtomi K; Kusagawa S; Taniguchi K; Shiino T; Kato A; Foongladda S;
Linkanonsakul S; Oka S I; Iwamoto A; Wasi C; Nagai Y; Takebe Y
 AIDS Research Center, National Institute of Infectious Diseases, Tokyo,
Japan.
 AIDS research and human retroviruses (UNITED STATES) Aug 10 1999, 15
(12) p1109-20, ISSN 0889-2229 Journal Code: 8709376
 Document type: Journal Article
 Languages: ENGLISH
 Main Citation Owner: NLM
 Record type: Completed
? t s3/3/2
 3/3/2
DIALOG(R)File 155:MEDLINE(R)
10391817   99383216   PMID: 10456313
 Accommodation of foreign genes into the Sendai virus genome: sizes of
inserted genes and viral replication.
 Sakai Y; Kiyotani K; Fukumura M; Asakawa M; Kato A; Shioda T; Yoshida T;
Tanaka A; Hasegawa M; Nagai Y
 Department of Viral Infection, Institute of Medical Science, University
of Tokyo, Japan.
 FEBS letters (NETHERLANDS) Aug 6 1999, 456 (2) p221-6, ISSN
0014-5793 Journal Code: 0155157
 Document type: Journal Article
 Languages: ENGLISH
 Main Citation Owner: NLM
 Record type: Completed
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    $2.98 Estimated total session cost   0.644 DialUnits
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File 155:MEDLINE(R) 1966-2002/Oct W2
*File 155: Alert feature enhanced for multiple files, duplicates
removal, customized scheduling. See HELP ALERT.

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        85465  VECTOR?
       524799  EXPRESSION
       892973  SYSTEM
         5908  EXPRESSION(W)SYSTEM
    S4    108  SENDAI AND (VECTOR? OR EXPRESSION(W)SYSTEM)
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    S5 1374088  PY>1999
? s s4 not s5
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      1374088  S5
    S6     78  S4 NOT S5
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 6/7/6
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10295835 99265151 PMID: 10332747
Gene delivery systems using the Sendai virus.
Nakanishi M; Mizuguchi H; Ashihara K; Senda T; Eguchi A; Watabe A; Nakanishi T; Kondo M; Nakagawa T; Masago A; Okabe J; Ueda S; Mayumi T; Hayakawa T
Department of Neurovirology, Osaka University, Japan. mahito@biken.osaka-u.ac.jp
Molecular membrane biology (ENGLAND) Jan-Mar 1999, 16 (1) p123-7, ISSN 0968-7688 Journal Code: 9430797
Document type: Journal Article; Review; Review, Tutorial
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Fusogenic liposome (FL) is a delivery system that can transfer encapsulated materials into living cells directly through membrane fusion. FL is a promising approach for gene therapy because it can deliver various genetic materials much more efficiently than other non-viral vectors without damaging the cell. FL-mediated gene transfer consists of two independent membrane fusion phenomena; generation of a FL by fusing a Sendai virus (SV) particle with a simple liposome encapsulating DNA, and successive fusion of the FL with cell membrane. The former requires viral F protein but no other special molecule on the liposomal membrane, whereas the latter may require the receptor (sialic acid) and unidentified assistant molecule(s) on the cell membrane. Further analysis suggests that these assistant molecule(s), not the receptor, may control the fusion and govern the cell specificity of FL-mediated delivery. This review has described a detailed analysis of these fusion phenomena and discussed possible applications of FL-mediated gene delivery to human gene therapy. (22 Refs.)
Record Date Created: 19990805

6/7/10
DIALOG(R)File 155:MEDLINE(R)
09999769 98412935 PMID: 9741904
Gene transfer vectors based on Sendai virus.
Nakanishi M; Mizuguchia H; Ashihara K; Senda T; Akuta T; Okabe J; Nagoshi E; Masago A; Eguchi A; Suzuki Y; Inokuchi H; Watabe A; Ueda S; Hayakawa T; Mayumi T
Department of Neurovirology, Research Institute for Microbial Diseases, Osaka University, Suita, Japan. mahito@biken.osaka-u.ac.jp
Journal of controlled release : official journal of the Controlled Release Society (NETHERLANDS) Jun 1998, 54 (1) p61-8, ISSN 0168-3659 Journal Code: 8607908
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
A gene delivery system is a fundamental technology used in human gene therapy. In order to treat patients suffering from incurable metabolic diseases, we must be able to deliver genes efficiently in situ and induce stable gene expression in non-dividing tissue cells. However, none of the current gene transfer systems (both viral and non-viral) satisfies this goal. In order to develop a novel gene delivery system that is free from the defects of existing gene transfer vectors, we analyzed natural biological phenomena that involve gene transfer and expression, and made artificial components that mimic the functioning of these systems. Our recent results shed light on three major aspects of gene transfer and expression: (1) the direct delivery of DNA into cytoplasm using fusogenic liposomes, (2) the transfer of DNA from cytoplasm to nucleus with a nuclear localization signal, and (3) the stabilization of DNA in the nucleus as an independent replicon. The possible development of a hybrid vector by combining these components is discussed.
Record Date Created: 19981118

6/7/11
DIALOG(R)File 155:MEDLINE(R)
09981932 98426248 PMID: 9751760
Site-specific gene delivery in vivo through engineered Sendai viral envelopes.
Ramani K; Hassan Q; Venkaiah B; Hasnain S E; Sarkar D P
Department of Biochemistry, University of Delhi South Campus, Benito Juarez Road, New Delhi-110021, India.
Proceedings of the National Academy of Sciences of the United States of America (UNITED STATES) Sep 29 1998, 95 (20) p11886-90, ISSN 0027-8424 Journal Code: 7505876
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Inspite of several stimulating developments in gene therapy, the formulation of a targeted gene delivery "vector" is still far from ideal. We have demonstrated the potential of reconstituted Sendai viral envelopes containing only the fusion glycoprotein (F-virosomes) in targeted delivery of reporter genes to liver cells of BALB/c mouse in vivo. The membrane fusion-mediated high efficiency of gene transfer to liver cells was ascertained following a critical evaluation of the level of the DNA, mRNA, and relevant proteins. Furthermore, the involvement of viral glycoprotein both as a unique natural ligand and as a membrane fusogen could lead to preferential transfection of parenchymal cell types of liver. The integration of transgenes in the mouse chromosomal DNA and its stable expression up to 4 mo after single i.v. administration of this gene carrier has bolstered its efficiency and novelty. Moreover, the F-virosomes did not elicit significant humoral immune response against the fusion protein in the injected animal. The findings reported here open up the possibility for

considering "F-virosomes" as a promising "vehicle" for site-specific DNA delivery in gene therapy.
Record Date Created: 19981022

6/7/15
DIALOG(R)File 155:MEDLINE(R)
09787921 98207469 PMID: 9545859
[Establishment of Sendai virus gene manipulation and its applications]
Kato A
Department of Viral Infection, University of Tokyo.
Uirusu. Journal of virology (JAPAN) Dec 1997, 47 (2) p133-44, ISSN 0042-6857 Journal Code: 0417475
Document type: Journal Article; Review; Review, Tutorial
Languages: JAPANESE
Main Citation Owner: NLM
Record type: Completed
(39 Refs.)
Record Date Created: 19980617

6/7/16
DIALOG(R)File 155:MEDLINE(R)
09766553 98200085 PMID: 9541016
Large quantity production with extreme convenience of human SDF-1alpha and SDF-1beta by a Sendai virus vector.
Moriya C; Shioda T; Tashiro K; Nagasawa T; Ikegawa M; Ohnishi Y; Kato A; Hu H; Xin X; Hasan M K; Maekawa M; Takebe Y; Sakai Y; Honjo T; Nagai Y
Department of Viral Infection, Institute of Medical Science, University of Tokyo, Japan.
FEBS letters (NETHERLANDS) Mar 20 1998, 425 (1) p105-11, ISSN 0014-5793 Journal Code: 0155157
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
We describe a robust expression of human stromal cell-derived factor-1alpha (SDF-1alpha) and SDF-1beta, the members of CXC-chemokine family, with a novel vector system based upon Sendai virus, a non-segmented negative strand RNA virus. Recombinant SDF-1alpha and SDF-1beta were detected as a major protein species in culture supernatants, reached as high as 10 microg/ml. This remarkable enrichment of the products allowed us to use even the crude supernatants as the source for biological and antiviral assays without further concentration nor purification and will thus greatly facilitate to screen their genetically engineered derivatives.
Record Date Created: 19980423

6/7/19
DIALOG(R)File 155:MEDLINE(R)
09605036 98031879 PMID: 9366551
Sendai virus-based expression of HIV-1 gp120: reinforcement by the V(-) version.
Yu D; Shioda T; Kato A; Hasan M K; Sakai Y; Nagai Y
Department of Viral Infection, Institute of Medical Science, University of Tokyo, Japan.
Genes to cells : devoted to molecular & cellular mechanisms (ENGLAND) Jul 1997, 2 (7) p457-66, ISSN 1356-9597 Journal Code: 9607379
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
BACKGROUND: We have established a system for recovering Sendai virus (SeV), a nonsegmented negative strand RNA virus, entirely from cDNA at an extremely high rate, and have succeeded in creating a V(-) SeV whose gene expression was greatly enhanced by the deletion of the nonessential V gene. Because of its extreme medical importance, there has been a strong need for the establishment of a better system to express the gp120 envelope glycoprotein of the human immunodeficiency virus type 1 (HIV-1) in sufficient quantity and purity. It also remains to be established to produce gp120 in in vitro natural host cells for HIV-1 such as human primary blood mononuclear cells, macrophages or established T cell lines. RESULTS: Using the above system, we created recombinant Sendai viruses expressing the gp120 in CV1 cells, a monkey kidney line. The expression level from the standard V(+) version has already reached 2.2/microg per 10(6) infected cells, which was readily purified from the culture fluid with a recovery rate of about 60%, and has so far appeared to be functionally and serologically authentic. The inserted gp120 gene was stably maintained during numerous passages of the recombinant virus. The V(-) version-based expression was even more robust, consistently reaching over 6.0 microg per 10(6) cells, a level that is one of the highest currently attainable for gp120 production in mammalian cells. Furthermore, a broad host range of SeV allowed gp120 production in all the three natural host cells for HIV-1 described above. CONCLUSIONS: SeV-based expression serves as a novel choice for producing large quantities of HIV-1 gp120 and will greatly facilitate biochemical, biological and immunological studies of this important glycoprotein.
Record Date Created: 19971230

6/7/20
DIALOG(R)File 155:MEDLINE(R)
09599670 98033190 PMID: 9367367
Creation of an infectious recombinant Sendai virus expressing the firefly luciferase gene from the 3' proximal first locus.
Hasan M K; Kato A; Shioda T; Sakai Y; Yu D; Nagai Y
Department of Viral Infection, Institute of Medical Science, University of Tokyo, Japan.

Journal of general virology (ENGLAND) Nov 1997, 78 (Pt 11) p2813-20,
ISSN 0022-1317 Journal Code: 0077340
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

A genetic engineering approach was made to generate a recombinant non-segmented negative-strand RNA virus, Sendai virus (SeV) of the family Paramyxoviridae, that expresses firefly luciferase. The DNA construct containing the entire open reading frame (ORF) of the luciferase gene followed by the SeV transcription stop and restart signals connected with the conserved intergenic three nucleotides was inserted immediately before the ORF of the viral 3'-proximal nucleocapsid (N) protein gene in a full-length SeV cDNA copy. After intracellular expression of full-length antigenomic transcripts from the engineered cDNA and of the viral n ucleocapsid protein and RNA polymerase from the respective plasmids, a recombinant SeV expressing luciferase activity at a high level was recovered, although the tendency of this particular reporter gene product to aggregate in cells made it difficult to estimate the maximum level of expression. The increase in genome length brought about by inserting 1728 nucleotides into the 15,384 nucleotide parental SeV was associated with reduced plaque size, slightly slower replication kinetics and a severalfold decrease in yield of the virus. The inserted luciferase gene was stably maintained after numerous rounds of replication by serial passages in chick embryos. These results indicate the potential utility of SeV as a novel expression vector.

Record Date Created: 19971208

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File 155:MEDLINE(R) 1966-2002/Oct W1
*File 155: Alert feature enhanced for multiple files, duplicates removal, customized scheduling. See HELP ALERT.

Set Items Description
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S2 1152747 DI OR DEFECTIVE(W)INTERFER?
S3 90 S1 AND S2
S4 99 DEFECTIVE AND S1
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4/7/30
DIALOG(R)File 155:MEDLINE(R)
08392662 95133230 PMID: 7831842
Measles virus nucleocapsid protein can function in Sendai virus defective interfering particle genome synthesis in vitro.
Chandrika R; Myers T; Moyer S A
Department of Molecular Genetics and Microbiology, University of Florida College of Medicine, Gainesville 32610.
Virology (UNITED STATES) Jan 10 1995, 206 (1) p777-82, ISSN 0042-6822 Journal Code: 0110674
Contract/Grant No.: AI 14594; AI; NIAID
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
The Sendai virus P and L proteins, the viral RNA polymerase, and the nucleocapsid protein, NP, synthesized in a transient mammalian expression system support the replication of Sendai virus defective interfering particle (DI) genome RNA in vitro. We have shown that the measles virus nucleocapsid protein, N, can substitute for the Sendai NP protein in genome synthesis. The chimeric product nucleocapsids, which contained Sendai RNA encapsidated with measles N protein, were atypical since they were sensitive to micrococcal nuclease digestion, unlike wild-type Sendai or measles nucleocapsids. The utilization of measles N protein required the endogenous Sendai virus RNA polymerase, since DI nucleocapsids free of polymerase were not replicated. Although both Sendai virus NP and P proteins and measles N and P proteins formed complexes when they were coexpressed, sedimentation analysis showed that measles N protein self-assembled and did not form a complex when expressed with the Sendai P protein. Furthermore, when the Sendai P-L polymerase complex was provided separately, measles N protein alone synthesized DI genome RNA in the absence of Sendai P protein. These data suggest that the self-assembled form of measles N protein functions in Sendai DI genome synthesis.
Record Date Created: 19950217

4/7/38
DIALOG(R)File 155:MEDLINE(R)
07796079 93323225 PMID: 8392616
The rule of six, a basic feature for efficient replication of Sendai virus defective interfering RNA.
Calain P; Roux L
Department of Genetics and Microbiology, University of Geneva Medical School, Switzerland.
Journal of virology (UNITED STATES) Aug 1993, 67 (8) p4822-30, ISSN 0022-538X Journal Code: 0113724
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
The addition of the hepatitis delta virus genomic ribozyme to the 3' end sequence of a Sendai virus defective interfering RNA (DI-H4) allowed the reproducible and efficient replication of this RNA by the viral functions expressed from cloned genes when the DI RNA was synthesized from plasmid. Limited nucleotide additions or deletions (+7 to -7 nucleotides) in the DI RNA sequence were then made at five different sites, and the different RNA derivatives were tested for their abilities to replicate. Efficient replication was observed only when the total nucleotide number was conserved, regardless of the modifications, or when the addition of a total of 6 nucleotides was made. The replicated RNAs were shown to be properly enveloped into virus particles. It is concluded that, to form a proper template for efficient replication, the Sendai virus RNA must contain a total number of nucleotides which is a multiple of 6. This was interpreted as the need for the nucleocapsid protein to contact exactly 6 nucleotides.
Record Date Created: 19930816

4/7/39
DIALOG(R)File 155:MEDLINE(R)
07617008 93139776 PMID: 8380835
Molecular cloning and characterization of a Sendai virus internal deletion defective RNA.
Engelhorn M; Stricker R; Roux L
Department of Genetics and Microbiology, University of Geneva Medical School, C.M.U., Switzerland.
Journal of general virology (ENGLAND) Jan 1993, 74 (Pt 1) p137-41,

ISSN 0022-1317 Journal Code: 0077340
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

A small defective Sendai virus RNA was selectively amplified from a virus preparation obtained after serial undiluted passages in embryonated eggs. Preliminary characterization showed that this defective RNA was a true internal deletion defective RNA, containing the 5' and 3' ends of the non-defective viral genomic RNA. Cloning of this RNA after reverse transcription and polymerase chain reaction amplification was performed in such a way that an exact copy of the defective RNA could be obtained by transcription of the plasmid with T7 RNA polymerase. Sequence analysis of the plasmid allowed further characterization of the defective RNA. It was shown potentially to encode a C-terminally truncated nucleocapsid (NP) protein of 162 amino acids. This truncated NP protein was identified in cells naturally infected with the defective virus preparation. Moreover the protein produced was shown to correspond to the protein synthesized in vitro from the T7 polymerase transcript of the cloned defective genome.

Record Date Created: 19930222

4/7/40
DIALOG(R)File 155:MEDLINE(R)
07508510 93033159 PMID: 1329337

Molecular cloning of natural paramyxovirus copy-back defective interfering RNAs and their expression from DNA.

Calain P; Curran J; Kolakofsky D; Roux L
Department of Genetics and Microbiology, University of Geneva Medical School, Switzerland.

Virology (UNITED STATES) Nov 1992, 191 (1) p62-71, ISSN 0042-6822 Journal Code: 0110674
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Using the unique sequence organization of copy-back defective interfering (DI) RNAs of paramyxoviruses, Sendai virus (SV), and measles virus copy-back DI RNAs were PCR amplified and cloned, without having to separate them from their helper nondefective genomes. The cloning was designed so that T7 polymerase transcription of the plasmids would generate DI RNAs with the exact 5' and 3' ends. The SV DI clone, transcribed from the plasmid in BHK cells using T7 polymerase produced by a vaccinia virus recombinant, was encapsidated and replicated by the SV-L, P/C, and NP proteins expressed from cloned genes. Such experiments open the possibility of examining the cis-acting sequences involved in viral multiplication directly, without using indirect markers such as CAT activity.

Record Date Created: 19921116

4/7/42
DIALOG(R)File 155:MEDLINE(R)
07399203 92333672 PMID: 1321276

Complexes of Sendai virus NP-P and P-L proteins are required for defective interfering particle genome replication in vitro.

Horikami S M; Curran J; Kolakofsky D; Moyer S A
Department of Immunology and Medical Microbiology, University of Florida, Gainesville 32610-0266.

Journal of virology (UNITED STATES) Aug 1992, 66 (8) p4901-8, ISSN 0022-538X Journal Code: 0113724
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

We present evidence that the formation of NP-P and P-L protein complexes is essential for replication of the genome of Sendai defective interfering (DI-H) virus in vitro, using extracts of cells expressing these viral proteins from plasmids. Optimal replication of DI-H nucleocapsid RNA required extracts of cells transfected with critical amounts and ratios of each of the plasmids and was three- to fivefold better than replication with a control extract prepared from a natural virus infection. Extracts in which NP and P proteins were coexpressed supported replication of the genome of purified DI-H virus which contained endogenous polymerase proteins, but extracts in which NP and P were expressed separately and then mixed were inactive. Similarly, the P and L proteins must be coexpressed for biological activity. The replication data thus suggest that two protein complexes, NP-P and P-L, are required for nucleocapsid RNA replication and that these complexes must form during or soon after synthesis of the proteins. Biochemical evidence in support of the formation of each complex includes coimmunoprecipitation of both proteins of each complex with an antibody specific for one component and cosedimentation of the subunits of each complex. We propose that the P-L complex serves as the RNA polymerase and NP-P is required for encapsidation of newly synthesized RNA.

Record Date Created: 19920814

4/7/44
DIALOG(R)File 155:MEDLINE(R)
06912656 91220648 PMID: 1850900

Rescue of a Sendai virus DI genome by other parainfluenza viruses: implications for genome replication.

Curran J A; Kolakofsky D
Department of Microbiology, University of Geneva School of Medicine, Switzerland.

Virology (UNITED STATES) May 1991, 182 (1) p168-76, ISSN 0042-6822 Journal Code: 0110674
Document type: Journal Article
Languages: ENGLISH

Main Citation Owner: NLM
Record type: Completed
Using a defective interfering Sendai virus stock (DIH4) freed of nondefective helper virus, we found that the closely related parainfluenza viruses 1 and 3 could substitute for the Sendai virus helper in replicating DIH4, creating chimeric nucleocapsids. The morbillivirus measles and the rhabdovirus VSV could not substitute. When DIH4 is incubated intracellularly for 5 days in the absence of help, the ability of PIV3 to rescue DIH4 at this time depended on fresh Sendai virus polymerase. The PIV3 polymerase apparently can only copy the chimeric template, but not that wrapped in the homologous Sendai NP protein. These results suggest that the cis-acting RNA sequences important for genome replication, e.g., the promoter and the encapsidation site, have been conserved among these viruses, but that the interactions between the polymerase and the template protein NP are unique for each virus.
Record Date Created: 19910604

4/7/48
DIALOG(R)File 155:MEDLINE(R)
06284543 89370318 PMID: 2549717
Replication of the genome RNAs of defective interfering particles of vesicular stomatitis and Sendai viruses using heterologous viral proteins.
Moyer S A
Department of Biochemistry and Molecular Biology, University of Florida College of Medicine, Gainesville 32610.
Virology (UNITED STATES) Sep 1989, 172 (1) p341-5, ISSN 0042-6822 Journal Code: 0110674
Contract/Grant No.: AI-14594; AI; NIAID
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
We have tested the ability of heterologous viral proteins to support the in vivo and in vitro replication of the RNA of defective interfering (DI) particles of two serotypes of VSV and of Sendai virus. In all the combinations of heterologous coinfections in vivo, DI particle replication was observed only in the coinfection with the VSV-Indiana DI particle and wild-type VSV-New Jersey. By quantitating RNA synthesis in reconstitution experiments we showed that with DI nucleocapsids isolated from infected cells, however, the soluble protein fraction from heterologous wild-type virus-infected cells could substitute in vitro to varying degrees for the homologous proteins in the elongation reaction of RNA replication and encapsidation. In these cases successful replication was confirmed by demonstrating the specific association of the heterologous N protein with the product nucleocapsid RNA. The initiation step, that is, the initial binding of the nucleocapsid protein to the leader RNA, in contrast, requires the homologous protein, since heterologous viral proteins could not support RNA replication and encapsidation from purified DI particles.
Record Date Created: 19890929

4/7/53
DIALOG(R)File 155:MEDLINE(R)
05719016 88128559 PMID: 2829428
Direct adverse effects of Sendai virus DI particles on virus budding and on M protein fate and stability.
Tuffereau C; Roux L
Microbiology Department, University of Geneva Medical School, Switzerland.
Virology (UNITED STATES) Feb 1988, 162 (2) p417-26, ISSN 0042-6822 Journal Code: 0110674
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Upon infections of BHK cells with a mixture of Sendai standard and defective interfering (DI) viruses (mixed virus infection), viral budding was found to be restricted by factors ranging from 5 to more than 20. The reduced viral budding correlated with a high intracellular M protein turnover. M appeared to be degraded shortly after its synthesis, and seemed not to be able to self-associate in a stable way under the plasma membrane as it did in St virus-infected cells. These data, added to the previous findings that infection with DI particles allowed infected cell survival and favored the cell-surface turnover of the hemagglutinin-neuraminidase protein, led to the hypothesis that DI genomes directly act by preventing the stable formation inside the cells of a viral structure composed of M/HN/nucleocapsids. When involved in this structure M would be protected from degradation and HN would be stably anchored in the plasma membrane. Formation of this structure would be necessary for viral budding and would be damaging for the cells. Comparison with results published by other authors shows that such a model is consistent with other data. It can integrate, as well, data obtained in the analysis of mutant viruses involved in persistence.
Record Date Created: 19880311

4/7/58
DIALOG(R)File 155:MEDLINE(R)
05567754 87321093 PMID: 2820122
Localization of functional sites on the hemagglutinin-neuraminidase glycoprotein of Sendai virus by sequence analysis of antigenic and temperature-sensitive mutants.
Thompson S D; Portner A
Virology (UNITED STATES) Sep 1987, 160 (1) p1-8, ISSN 0042-6822 Journal Code: 0110674
Contract/Grant No.: AI-11949; AI; NIAID; CA21765; CA; NCI

Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

To locate the various functions associated with the hemagglutinin-neuraminidase (HN) glycoprotein of Sendai virus in the primary structure of the protein, a temperature-sensitive (ts) mutant and seven antigenic mutants were sequenced. The ts mutant was defective in its ability to agglutinate erythrocytes and infect host cells, while its neuraminidase activity was normal. Its sequence revealed two closely spaced amino acid substitutions (residues 262 and 264) and one distant substitution (residue 461). Revertants could not be isolated, suggesting that more than one of the substitutions is responsible for the defective hemagglutinating activity. The antigenic mutants were selected with monoclonal antibodies that delineate four nonoverlapping antigenic sites (I-IV) and separately inhibit hemagglutinating, neuraminidase, and hemolysis activities. Mutants selected with antibodies to antigenic sites I-III were used to map these functions on the primary sequence of HN. Each antigenic mutant had a single point mutation in the HN gene that resulted in an amino acid substitution in the protein. A site II mutant selected with an antibody which inhibits hemolysin activity had a substitution at amino acid 420, while a mutant selected with antibody that inhibits only erythrocyte binding (site III) had a substitution at amino acid 541. Two antigenic mutants selected with an antibody that inhibits hemagglutination and neuraminidase activities (site I) had amino acid substitutions in close proximity (residues 277 and 279) to the two closely spaced substitutions of the ts mutant. These findings suggest that the region defined by the ts mutant and these two antigenic mutants is involved in host cell binding. Antigenic mutants selected with another site I antibody had amino acid changes at residue 184, indicating that antigenic site I is discontinuous in the primary sequence. This antibody blocks only hemagglutination, but mutants selected with it had a decreased neuraminidase activity. This finding supports the idea that the neuraminidase site is close to, but distinct from, the hemagglutination site.

Record Date Created: 19871014

4/7/59
DIALOG(R)File 155:MEDLINE(R)
05463074 87209507 PMID: 2883862

Infection of the central nervous system of mice by standard Sendai virus, defective interfering Sendai virus and the mixture of both: comparison of virus multiplication and pathogenicity.

Ruttkay-Nedecka S; Rajcani J; Eleckova E; Ruttkay-Nedecky G

Acta virologica. English ed (CZECHOSLOVAKIA) Jan 1987, 31 (1) p78-82 , ISSN 0001-723X Journal Code: 0370401

Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

The intracerebral (i.c.) infection of newborn mice with standard Sendai virus (SV), defective interfering Sendai virus (DV) and their mixture (SV + DV) has been used as a model for the possible role of defective interfering particles of paramyxoviruses in several chronic degenerative diseases of central nervous system (CNS). The dynamics of Sendai virus multiplication and virus distribution in CNS of mice, as well as the histological changes and the clinical symptoms were evaluated for up to 112 days post-infection (p.i.). The infectious virus was detected in the brains of animals inoculated i.c. either with SV, or DV, or SV + DV as soon as by 5 hr p.i., with maximum infectivity titre at 24 hr p.i. In brains of animals inoculated with SV, the virus was detected until 5th day p.i.; nevertheless in those, inoculated with SV + DV or DV, low infectious titres could be detected even at later intervals. In mice inoculated i.c. with DV, traces of Sendai virus were detected in subpassages, as late as 3 months p.i.

Record Date Created: 19870608

4/7/60
DIALOG(R)File 155:MEDLINE(R)
05192610 86265167 PMID: 2873735

Purification and characterization of defective interfering particles of Sendai virus.

Ruttkay-Nedecka S

Acta virologica. English ed (CZECHOSLOVAKIA) Mar 1986, 30 (2) p170, ISSN 0001-723X Journal Code: 0370401

Document type: Letter
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Record Date Created: 19860730

4/7/62
DIALOG(R)File 155:MEDLINE(R)
05125596 86200404 PMID: 3009869

Nucleotide sequences that affect replicative and transcriptional efficiencies of Sendai virus deletion mutants.

Re G G; Kingsbury D W

Journal of virology (UNITED STATES) May 1986, 58 (2) p578-82, ISSN 0022-538X Journal Code: 0113724

Contract/Grant No.: AI 05343; AI; NIAID; CA 21765; CA; NCI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Structural features of the genomes of virus deletion mutants (DI virions) influence their replication efficiency. Among nonsegmented negative-strand

RNA viruses, substitution of the genomic 3' terminus by a complementary copy of the 5' terminus (so-called "copy-back" sequence) could enhance replication either because the new 3' end is a better promoter of RNA replication or because DI RNAs that possess this sequence are incapable of acting as templates for transcription. Here we provide evidence that both mechanisms operate in mixed infections with Sendai virus DI RNAs. RNAs incapable of transcription always outgrew RNA species that were transcribed. This was true even when the 3'-terminal sequence of the untranscribed RNA was identical to the genomic 3' terminus, as in the case of an internally deleted DI genome (RNA Ra) rendered transcriptionally inert by point mutations of bases 47 and 51 at the 5' end of the positive-strand leader RNA template. Nevertheless, Ra was outgrown by a copy-back DI RNA, indicating that the 3' genomic end of Ra is a less efficient site for replication initiation than the copy-back sequence.
Record Date Created: 19860527

4/7/63
DIALOG(R)File 155:MEDLINE(R)
04924853 85301972 PMID: 2994295
Expression of Sendai virus defective-interfering genomes with internal deletions.
Hsu C H; Re G G; Gupta K C; Portner A; Kingsbury D W
Virology (UNITED STATES) Oct 15 1985, 146 (1) p38-49, ISSN 0042-6822 Journal Code: 0110674
Contract/Grant No.: AI 05343; AI; NIAID; AI 11949; AI; NIAID; CA 21765; CA; NCI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Sendai virus strain 7 has been shown to contain four defective interfering (DI) RNA species in which both genome termini and various adjacent fragments of the 3'-terminal NP gene and 5'-terminal L gene are represented, but most or all internal genes and gene boundaries are deleted. Previous sequence analyses of these mutant RNAs suggested that all four possessed the transcription initiation signal of the NP gene and the transcription termination signal of the L gene. The supposition that these signals should specify transcripts has now been supported by oligo(dT) selection of four DI 7 specific RNA species that had apparent molecular weights slightly lower than each DI genome. DI RNA 7a, which contains the entire NP gene, except for two U residues at the end of the poly(A) initiation signal, appeared to be transcribed solely as a readthrough product. Since DI RNA 7a contains the entire NP protein-coding sequence and DI RNAs 7c and 7d contain fragments of it, whereas DI RNA 7b is devoid of it, only transcripts of RNAs 7c and 7d were expected to specify fusion proteins containing NP gene-specific sequences. A strain 7-induced protein that reacted with monoclonal antibodies against the NP protein had the 33,000 Mr size appropriate for the translation product predicted by the sequence of RNA 7d. Other proteins of lower molecular weight were seen only in cells infected by strain 7, but they did not react with NP-specific antibody and their translation in vitro was not blocked by hybridization to an NP gene-specific oligonucleotide. Therefore, at least some of these proteins may be cellular products induced by DI virus infection. These DI transcripts and translation products may influence interference with replication of the parental helper virus.
Record Date Created: 19851016

4/7/64
DIALOG(R)File 155:MEDLINE(R)
04924852 85301971 PMID: 2994294
Nucleotide sequences responsible for generation of internally deleted Sendai virus defective interfering genomes.
Re G G; Morgan E M; Kingsbury D W
Virology (UNITED STATES) Oct 15 1985, 146 (1) p27-37, ISSN 0042-6822 Journal Code: 0110674
Contract/Grant No.: AI 05343; AI; NIAID; CA 21765; CA; NCI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
The deletion points of four internally deleted defective interfering (DI) RNA species (7a, 7b, 7c, and 7d) that reside in a single Sendai virus strain were defined by nucleotide sequencing. DI RNA 7a (Mr 1.24 x 10(6)) retained the entire NP gene with the complete NP protein-coding sequence, except for the last two U residues of the polyadenylation signal, fused to an 1800-nucleotide sequence comprising 5'-terminal genome and adjacent L gene sequences. DI RNA 7b (Mr, 0.70 x 10(6)) consisted of 100 3'-terminal nucleotides fused to 1900 5'-terminal bases; the deletion point in the NP gene precedes the NP protein initiation codon. DI RNA 7c (Mr 0.55 x 10(6)) retained 420 3'-terminal and 1150 5'-terminal nucleotides. The sequence just downstream of the sequenced deletion site is M gene specific, indicating that 7c arose from at least two deletion events and that it comprises NP, M, and L gene fragments. Transcription of RNA 7c could yield an MRNA encoding a fusion protein with a 14,000 Mr (N-terminal NP sequence fused to out of frame M-specific amino acids). DI RNA 7d (Mr 0.92 x 10(6)) retained 1027 3'-terminal nucleotides fused to 1600 bases from the 5'-terminus. It has an open reading frame for a 33,000 Mr N-terminal NP protein fragment. Nucleotide sequences flanking each deletion and just downstream of the NP gene deletion site suggested that these DI genomes were generated by a copy-choice mechanism, involving polymerase jumping during replication of negative polarity virus genome templates. In this process, the termination and reinitiation of RNA synthesis would involve recognition of sequences that regulate virus genome transcription and replication.

Record Date Created: 19851016

4/7/65
DIALOG(R)File 155:MEDLINE(R)
04823677 85209814 PMID: 2987404
Three variations in the cell surface expression of the haemagglutinin-neuraminidase glycoprotein of Sendai virus.
Roux L; Beffy P; Portner A
Journal of general virology (ENGLAND) May 1985, 66 (Pt 5) p987-1000, ISSN 0022-1317 Journal Code: 0077340
Contract/Grant No.: AI-11941; AI; NIAID; CA 21765; CA; NCI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
The fate of the haemagglutinin-neuraminidase glycoprotein (HN) of Sendai virus in three types of infection was studied by measuring its sensitivity to endoglycosidase H and its rate of appearance and turnover at the cell surface. HN behaved differently in the three types of infection. When highly expressed at the surface, as in a lytic standard virus infection, HN accumulated at the surface in a stable form (half-life of disappearance from the surface much greater than 10 h). When moderately expressed, as in a non-lytic standard virus plus defective interfering virus infection, HN reached the membrane normally, but turned over rapidly (half-life about 2 h) and was re-internalized. When poorly expressed, as in long-term persistent infection, HN did not reach the cell surface and appeared to be degraded before reaching it. In contrast to HN, the other viral glycoprotein, F0, exhibited a similar turnover rate at the cell surface in the three situations. However, when compared to surface expression in standard virus-infected cells under standardized conditions, F0 surface expression in persistently infected cells was reduced. This reduction correlates with a decreased maturation rate in these cells.
Record Date Created: 19850705

4/7/67
DIALOG(R)File 155:MEDLINE(R)
04807641 85185678 PMID: 2985811
In vitro replication of Sendai virus wild-type and defective interfering particle genome RNAs.
Carlsen S R; Peluso R W; Moyer S A
Journal of virology (UNITED STATES) May 1985, 54 (2) p493-500, ISSN 0022-538X Journal Code: 0113724
Contract/Grant No.: AI14594; AI; NIAID
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
A system for studying the in vitro replication of the genome RNAs of Sendai virus and its defective interfering particle DI-H has been developed. Cytoplasmic extracts of baby hamster kidney cells infected with wild-type Sendai virus or coinfected with wild-type Sendai virus plus DI-H were prepared after lysolecithin treatment at 12 h postinfection. The extracts supported the transcription of six viral mRNAs as well as the replication of the Sendai virus 50S (wild-type) and 14S DI-H genome RNAs and their encapsidation into nucleocapsids in the absence of de novo protein synthesis. RNA replication in vitro represented more than 50% of total RNA synthesis, a relative level higher than that found in the infected cell. The proteins required for Sendai virus RNA replication were present in a soluble protein pool at the time of extract preparation. Depletion of the protein pool by prior treatment of infected cells with cycloheximide inhibited subsequent in vitro genome replication without affecting transcription. The cytoplasmic extract may be separated by high-speed centrifugation into two components: the Sendai virus wild-type and DI-H nucleocapsid templates containing the RNA and associated NP, L, and P proteins and the soluble protein fraction containing primarily the P, NP, and M viral proteins with trace amounts of the L, HN, Fo, and nonstructural C proteins. The isolated intracellular DI-H nucleocapsid template alone cannot replicate its RNA, but when recombined with the Sendai virus soluble protein fraction it catalyzes the replication and encapsidation of viral RNAs. The initiation of RNA replication in vitro can be demonstrated because detergent-disrupted purified DI-H virions replicate both positive- and negative-strand RNAs in the presence, but not in the absence, of the soluble protein fraction from an extract of infected cells.
Record Date Created: 19850529
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Temp SearchSave "TD772" stored
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$0.00 99 Type(s) in Format 6
$3.36 16 Type(s) in Format 7
$3.36 115 Types
$6.93 Estimated cost File155
$2.81 TELNET
$9.74 Estimated cost this search
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$0.01 TYMNET
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*File 155: File temporarily is not updating. The updating will resume by the end of January 2002.

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S1	2677	SENDAI
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	1574	NONESSENTIAL
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	155055	ESSENTIAL
	970	NON(W)ESSENTIAL
	2677	S1
S2	9	(NONESSENTIAL OR NON(W)ESSENTIAL) AND S1

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2/7/5
DIALOG(R)File 155:MEDLINE(R)
10216733 99314114 PMID: 10386336
Paramyxovirus replication and pathogenesis. Reverse genetics transforms understanding.
Nagai Y
Department of Viral Infection, University of Tokyo, Japan.
Reviews in medical virology (ENGLAND) Apr-Jun 1999, 9 (2) p83-99, ISSN 1052-9276 Journal Code: DET
Languages: ENGLISH
Document type: Journal Article; Review; Review, Tutorial
Record type: Completed
A recent breakthrough in the field of nonsegmented negative strand RNA viruses (Mononegavirales), including paramyxoviruses, is the establishment of a system to recover an infectious virus entirely from complementary DNA and hence allow reverse genetics. Mutations can now be introduced into viral genomes at will and the resulting phenotypes studied as long as the introduced mutations are not lethal. This technology is being successfully applied to answer outstanding questions regarding the roles of viral components in replication and their contribution to pathogenicity, which are difficult to address using conventional virology. For instance, how the paramyxovirus accessory proteins V and C contribute to actual viral replication and pathogenesis has remained unanswered since their first description more than 20 years ago. Using Sendai virus, which causes fatal pneumonia in mice, it has been shown that the V protein is completely dispensable for viral replication in cell cultures but encodes a luxury function required for pathogenesis in vivo. The Sendai virus C proteins were also defined to be nonessential gene products which greatly contributed to replication both in vitro and in vivo. It is also now possible to design live vaccines by introducing predetermined or plausible attenuating mutations. In addition, the use of paramyxoviruses to express foreign genes has also become feasible. Paramyxovirus reverse genetics is thus renovating our understanding of viral replication and pathogenesis and will further mark an era in recombinant technology for disease prevention and gene therapy. (95 Refs.)
Record Date Created: 19990819

2/7/6
DIALOG(R)File 155:MEDLINE(R)
09780672 98266442 PMID: 9605405
Sendai virus C proteins are categorically nonessential gene products but silencing their expression severely impairs viral replication and pathogenesis.
Kurotani A; Kiyotani K; Kato A; Shioda T; Sakai Y; Mizumoto K; Yoshida T; Nagai Y
Department of Viral Infection, Institute of Medical Science, University of Tokyo, Japan.
Genes to cells (ENGLAND) Feb 1998, 3 (2) p111-24, ISSN 1356-9597 Journal Code: CUF
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
BACKGROUND: The P/C mRNA of Sendai virus (SeV), a prototypic member of the family Paramyxoviridae in the Mononegavirales superfamily comprising a large number of nonsegmented negative strand RNA viruses, encodes a nested set of accessory proteins, C', C, Y1 and Y2, referred to collectively as C proteins, initiating, respectively, at ACG/81 and AUGs/114, 183, 201 in the +1 frame relative to the ORF of phospho (P) protein, the smaller subunit of RNA polymerase. Among them, C is the major species expressed in infected cells at a molar ratio which is several-fold higher than the other three. However, their function has remained an enigma. It has not even been established whether or not the C proteins are essential for viral replication. Many other viruses in Mononegavirales encode C-like proteins, but their roles also remain to be defined. RESULTS: By taking advantage of a recently developed reverse genetics system to recover infectious SeV from cDNA, we created mutants in which C protein frames were variously silenced. C/C'(-) viruses which did not express C and C', but did express Y1 and Y2, were severely attenuated in replication in tissue culture cells of various species and tissues, as well as in embryonated chicken eggs. More notably,

they were almost totally incapable of growing productively in--and hence nonpathogenic for mice--the natural host. Both gene expression and genome replication appeared to be impaired in C/C'(-) viruses. Additionally silencing the Y1 and Y2 expression was also possible, and a critically impaired but viable clone, the 4C(-) virus, was isolated which expressed none of the four C proteins. CONCLUSION: SeV C proteins are categorically nonessential gene products, but greatly contribute to full replication capability in vitro and are indispensable for in vivo multiplication and pathogenesis. This study represents the first comprehensive functional assessment of the accessary C protein for Mononegavirales.
Record Date Created: 19980709

2/7/7
DIALOG(R)File 155:MEDLINE(R)
09597432 98031879 PMID: 9366551
Sendai virus-based expression of HIV-1 gp120: reinforcement by the V(-) version.
Yu D; Shioda T; Kato A; Hasan MK; Sakai Y; Nagai Y
Department of Viral Infection, Institute of Medical Science, University of Tokyo, Japan.
Genes to cells (ENGLAND) Jul 1997, 2 (7) p457-66, ISSN 1356-9597 Journal Code: CUF
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
BACKGROUND: We have established a system for recovering Sendai virus (SeV), a nonsegmented negative strand RNA virus, entirely from cDNA at an extremely high rate, and have succeeded in creating a V(-) SeV whose gene expression was greatly enhanced by the deletion of the nonessential V gene. Because of its extreme medical importance, there has been a strong need for the establishment of a better system to express the gp120 envelope glycoprotein of the human immunodeficiency virus type 1 (HIV-1) in sufficient quantity and purity. It also remains to be established to produce gp120 in in vitro natural host cells for HIV-1 such as human primary blood mononuclear cells, macrophages or established T cell lines. RESULTS: Using the above system, we created recombinant Sendai viruses expressing the gp120 in CV1 cells, a monkey kidney line. The expression level from the standard V(+) version has already reached 2.2/microg per 10(6) infected cells, which was readily purified from the culture fluid with a recovery rate of about 60%, and has so far appeared to be functionally and serologically authentic. The inserted gp120 gene was stably maintained during numerous passages of the recombinant virus. The V(-) version-based expression was even more robust, consistently reaching over 6.0 microg per 10(6) cells, a level that is one of the highest currently attainable for gp120 production in mammalian cells. Furthermore, a broad host range of SeV allowed gp120 production in all the three natural host cells for HIV-1 described above. CONCLUSIONS: SeV-based expression serves as a novel choice for producing large quantities of HIV-1 gp120 and will greatly facilitate biochemical, biological and immunological studies of this important glycoprotein.
Record Date Created: 19971230

2/7/8
DIALOG(R)File 155:MEDLINE(R)
09526206 97186724 PMID: 9034340
The paramyxovirus, Sendai virus, V protein encodes a luxury function required for viral pathogenesis.
Kato A; Kiyotani K; Sakai Y; Yoshida T; Nagai Y
Department of Viral Infection, Institute of Medical Science, University of Tokyo, Minato-ku, Japan.
EMBO journal (ENGLAND) Feb 3 1997, 16 (3) p578-87, ISSN 0261-4189 Journal Code: EMB
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
The Sendai virus (SeV) V protein is characterized by the unique cysteine-rich domain in its carboxy-terminal half which is fused to the amino-terminal half of the P protein, but its function has remained enigmatic. The V protein-directing mRNA is generated by a remarkable process known as mRNA editing involving the pseudotemplated addition of a single G residue at a specific septinucleotide locus in the P gene, whereas the unedited exact copy encodes the P protein. Here, we introduced two nucleotide changes in the septinucleotide motif (UUUUCCC to UUCUUCC) in a full-length SeV cDNA and were able to recover a virus from the cDNA, which was devoid of mRNA editing and hence unable to synthesize the V protein. Compared with the parental wild-type virus with regard to gene expression, replication and cytopathogenicity in various cell lines in vitro, the V(-) virus was found to be either potentiated or comparable but never attenuated. The V(-) virus, however, showed markedly attenuated in vivo replication capacity in and pathogenicity for mice. Thus, though categorized as a nonessential gene product, SeV V protein encodes a luxury function required for in vivo pathogenicity.
Record Date Created: 19970313

2/7/9
DIALOG(R)File 155:MEDLINE(R)
07168996 92007754 PMID: 1655410
The Sendai virus P gene expresses both an essential protein and an inhibitor of RNA synthesis by shuffling modules via mRNA editing.
Curran J; Boeck R; Kolakofsky D
Department of Genetics and Microbiology, University of Geneva, School of Medicine, Switzerland.
EMBO journal (ENGLAND) Oct 1991, 10 (10) p3079-85, ISSN 0261-4189 Journal Code: EMB

Languages: ENGLISH
Document type: Journal Article
Record type: Completed

The P gene of Sendai virus expresses as many as eight proteins, two of which (V and W) are expressed only from edited mRNAs; only the P protein is known to be involved in RNA synthesis. To examine the functions of the other P gene proteins, we developed an in vivo system in which genome replication is driven by plasmid generated viral proteins. We found that P was essential for this process, whereas V and W were not only non-essential, they were inhibitory. By using various P gene deletions and varying the amounts of plasmids transfected, we provide evidence that P is a modular protein. The N-terminal domain (shared with V and W) binds the L or polymerase protein, whereas the C-terminal domain binds the nucleoprotein NP. A model of paramyxovirus RNA synthesis is presented, and the implications of negative regulation during persistent infection are discussed.

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DIALINDEX(R)
  (c) 2002 The Dialog Corporation plc
*** DIALINDEX search results display in an abbreviated ***
*** format unless you enter the SET DETAIL ON command. ***
? sf allscience
  You have 260 files in your file list.
  (To see banners, use SHOW FILES command)
? s sendai and au=kato? and py=1996

Your SELECT statement is:
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           2   34: SciSearch(R) Cited Ref Sci_1990-2002/Jan W3
           1   47: Gale Group Magazine DB(TM)_1959-2002/Jan 16
           1   50: CAB Abstracts_1972-2002/Dec
           1   71: ELSEVIER BIOBASE_1994-2002/Jan W2
           1   73: EMBASE_1974-2002/Jan W2
           1   76: Life Sciences Collection_1982-2002/Jan
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ZEXEL
      CORP HIG
           1  348: EUROPEAN PATENTS_1978-2002/Jan W03
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           3  440: Current Contents Search(R)_1990-2002/Jan W3
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  16 files have one or more items; file list includes 260 files.
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File 348:EUROPEAN PATENTS  1978-2002/Jan W03
     (c) 2002 European Patent Office

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     (c) 2002 European Patent Office

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? t s2/ti/1

2/TI/1
DIALOG(R)File 348:(c) 2002 European Patent Office. All rts. reserv.
Flat tube for heat exchanger and method for producing same
Flachrohr fur Warmetauscher und Verfahren zu dessen Herstellung
Tube plat pour echangeur de chaleur et son procede de fabrication
? file 155;exs
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File 155:MEDLINE(R) 1966-2002/JAN W3
*File 155: File temporarily is not updating. The updating will
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Initiation of Sendai virus multiplication from transfected cDNA or RNA
with negative or positive sense.
Kato A; Sakai Y; Shioda T; Kondo T; Nakanishi M; Nagai Y
Department of Viral Infection, University of Tokyo, Japan.
Genes to cells (ENGLAND) Jun 1996, 1 (6) p569-79, ISSN 1356-9597
Journal Code: CUF
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
BACKGROUND: The mononegavirus superfamily (Mononegavirales) comprises three families, Rhabdoviridae, Paramyxoviridae and Filoviridae. These viruses possess a single stranded negative sense RNA as the genome. Recent success in the recovery of infectious virus from a transfected cDNA of mononegaviruses including Sendai virus, a prototypic paramyxovirus, is opening the possibility of their genetic engineering. However, infectious viruses have been recovered only by initiating the infectious cycle with cDNA directing the synthesis of antigenomic positive sense (+)RNA. Starting with genomic negative sense (-)RNA has been unsuccessful. Furthermore, the recovery efficiency has often been extremely low. RESULTS: We describe here an analogous system that allows recovery of Sendai virus at a high rate, from cells in which the transfected cDNA and plasmids to support the synthesis of viral nucleocapsid protein and RNA polymerases are coexpressed by vaccinia virus-driven bacteriophage T7 polymerase. Our system was able to recover the virus from cDNA directing not only (+)RNA but also (-)RNA. Moreover, using this system, we succeeded in recovery of the virus by transfection of in vitro synthesized (+)RNA or (-)RNA. This improved virus recovery appeared to be accomplished by supplying the supporting plasmids at an optimal ratio and by minimizing the cytopathic effect of the vaccinia virus by specific inhibitors. In addition, it was probably critical that our cDNAs were constructed to generate viral authentic RNAs without adding T7 promoter-specific nucleotides to the 5' ends. An immediate application of the system was demonstrated by the creation of a candidate vaccine strain with a predetermined attenuating mutation in the cleavage-activation site of the viral fusion glycoprotein. CONCLUSION: We have established methods which greatly improve the recovery of Sendai virus from cDNA. There is essentially no absolute obstacle to recovery of the virus from the (-)RNA template. Even the complete full length RNA chain in the naked form appears to be properly encapsidated to become a functional template.
Record Date Created: 19970425
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